Exhibit B

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REVIEW

FRONTAL LOBE CHANGES IN ALCOHOLISM: A REVIEW OF THE LITERATURE

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Abstract — Alcohol can induce a wide spectrum of effects on the central nervous system. These effects can be recognized at the neurophysiological, morphological and neuropsychological levels. Several studies of the effect of alcohol on the frontal lobes were identified for review from MedLine, PsychLIT databases and by manual searching. In this review article, the different changes are examined in detail. Computed tomography studies have reported changes of frontal lobe in alcoholism, while magnetic resonance imaging studies supported these findings. Neurophysiological studies with positron emission tomography and single photon emission computed tomography have reported a decreased frontal lobe glucose utilization and reduced cerebral blood flow. There is also evidence from neuropsychological studies that there are specific deficits in alcoholism that suggest frontal lobe dysfunction. Considered together, these studies lend a strong credence to the concept of frontal lobe pathology in alcoholism. However, frontal lobe is not an isolated part of the brain and should be considered with its heavy connections to different cortical and subcortical areas of the brain.

INTRODUCTION

In a number of ways, the study of the frontal lobes might be described as the study of the qualities that differentiate a human being from other animals. In 1928 the American neurologist Tilney suggested that the entire period of human evolutionary existence could be considered the 'age of the frontal lobe'. A great deal of indirect evidence supported such a claim (Stuss and Benson, 1984).

Current literature emphasized a primary hypothesis concerning the nature and extent of frontal lobe involvement in alcoholism. Considerable research has focused on the pattern of anterior brain deficit. Tarter (1975*a*) has suggested that prolonged alcohol abuse results in effects that are most pronounced in the anterior region of the brain, extending from the frontal lobe through the dorsomedial nucleus of the thalamus and associated basal regions.

This review will cover the current state of knowledge of frontal lobe dysfunction in alcoholism. The frontal lobe dysfunction will be presented with a major focus on changes that occur in adult humans. References to the animal literature will be limited, except for the sections on neuroanatomy and neurophysiology, where non-human data are the basis.

NEUROANATOMICAL CONSIDERATIONS

Anatomically, the frontal lobes are the massive cerebral area anterior to the rolandic fissure and above the sylvian fissure. There are two roughly symmetrical lobes, each of which can be divided into three areas: dorsal–lateral, medial, and basilar–orbital. Actually, the frontal lobe may be divided in any number of different ways (Stuss and Benson, 1984).

Frontal lobes compose the single largest cortical region in the brain. The prefrontal cortex is the most complex and highly developed of the neocortical regions in the human brain. Functioning as a massive association cortex, it has afferent and efferent connections to all other neocortical regions i.e. parietal, temporal and occipital, as well as to cingulate, limbic, and basal ganglia structures. The thalamus serves as a major junctional complex that modulates input to the prefrontal cortex, and it has been proposed that the prefrontal cortex should be defined on the basis of its anatomical relationship with the medial dorsal thalamic nucleus (Nauta, 1971, 1972).

The importance of the frontal lobes derives from rich connections, both afferent and efferent, with almost all other parts of the central nervous system. Frontal connections with cortical sensory areas, providing information from the external milieu, occur either by direct cortical–cortical afferents or via the thalamus. The occipital, parietal, and temporal sensory association cortices connect to both the anterior temporal and inferior parietal areas; in turn, each of these has direct afferent connections to the frontal cortex. The prefrontal cortex receives projections from olfactory sensation; it is thus the only cortical area interacting with all four sensory modalities. The frontal lobe also has well-developed connections with limbic and subcortical areas that provide monitoring of the internal milieu (Nauta, 1971, 1972).

The prefrontal cortex is the single largest brain region in human beings, having been estimated to constitute 29% of the total cortex (Nauta, 1971; Goldman-Rakic *et al.*, 1984; Fuster, 1986; Stuss and Benson, 1986) (see Figs 1 and 2).

FUNCTIONAL CONSIDERATIONS

The specific functions of the different frontal lobe regions are still in the process of being mapped. Studies of experimental lesions in animals and traumatic or disease-induced lesions in humans have indicated that injury to the prefrontal cortex leads to disorders of categorizing (Andreasen *et al.*, 1986). Furthermore, a decrease in voluntary motor behaviour,

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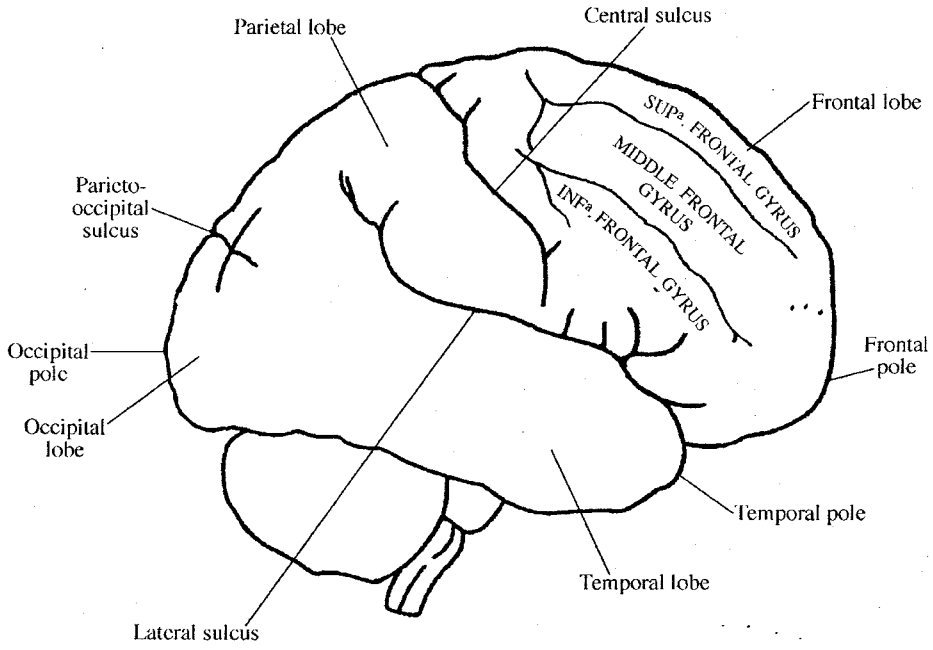

Fig. 1. Cerebral cortex viewed from its right lateral aspect, showing the lobes and large sulci.

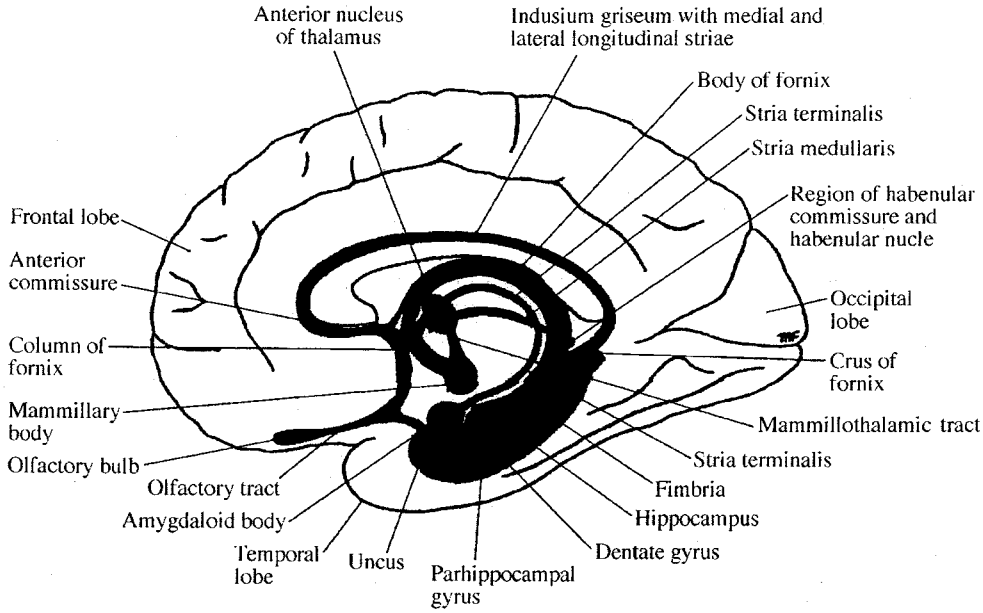

Fig. 2. Medial aspect of right cerebral hemisphere, showing structures that form the limbic system and medial aspect of temporal lobe connects to frontal lobe.

decreased will and energy, a tendency to engage in repetitive or preservative behaviour, difficulty in shifting response set, and abnormalities of affect and emotion, particularly apathy, indifference, and shallowness also occur (Jacobson, 1935; Hebb, 1945; Nauta, 1964, 1971; Drewe, 1975; Damasio, 1979). Other symptoms consistent with dysfunction of the frontal lobes can include problems with short-term memory, planning, problem solving, impulsivity, disinhibition, and poor motivation (Krans and Maki, 1997). The frontal lobe is involved in functions such as creative thinking, planning of future actions, decision making, artistic expression, aspects of emotional behaviour, as well as spatial working memory,

language and motor control (Miotto *et al.*, 1996; Semendeferi *et al.*, 1997), sustaining attention over time (Rueckert and Grafman, 1996), and smooth pursuit eye movement (Heide *et al.*, 1996).

Efforts have been made to understand frontal lobe deficits in terms of cognitive psychology and artificial intelligence theory (Shallice and Evans, 1978; Shallice, 1982). Shallice (1982) postulated two separate operations in problem solving. Contention scheduling is the term used to describe the fast, efficient, routine use of limited resources. With the introduction of non-routine factors (e.g. a new problem), a general programming, planning, and monitoring system, the supervisory attentional system (SAS) is made operational (Stuss and Benson, 1984). Shallice's theory appears to fit many examples from daily life. For routine tasks, contention scheduling is adequate. Thus a person may drive home and not truly be aware of his behaviour. The SAS can rest or deal with other information, and contention schemas correctly handle the routine behaviours. 'Capture errors' may occur (Norman, 1981). A strong trigger might activate contention scheduling and lead to incorrect response. The SAS, not monitoring the routine behaviour, would be unaware until later (Stuss and Benson, 1986).

PHYSIOLOGICAL DYSFUNCTION OF FRONTAL LOBE IN ALCOHOLISM

Positron emission tomography (PET) studies

Mapping the functional activity of the brain is just one of the current applications of PET. In this context, functional anatomy is described as the relationship between the topography of neural activity and cognitive behavioural or psychomotor tests. The basic principle behind functional mapping is the matching of neuronal activity to motor states, psychological states or pathological states (Dolan, 1992). PET uses as tracers compounds labelled with short-lived positron-emitting isotopes. By tomographically recording their distribution in the body, PET allows the assessment of parameters of tissue function regionally, quantitatively and non-invasively. Two early studies have reported measurements of regional cerebral glucose metabolism in chronic alcoholics. One report found normal absolute values of metabolic rates of glucose in all regions, but a significantly reduced regional distribution index (ratio between regional value and mean cortical value) was found in the medial frontal cortex (Samson *et al.*, 1986). The second study found significantly lower cerebral metabolic rates of glucose in the alcoholic group, than in normal controls; in addition, the alcoholics had fewer significant interregional correlations and also failed to show the normal response of increasing right hemisphere glucose metabolism following a non-verbal auditory stimulus (Sachs *et al.*, 1987).

Later literature revealed decreased local cerebral metabolic rates for glucose bilaterally in the medial–frontal area of the cerebral cortex in alcohol-dependent patients (Gilman *et al.*, 1990; Adams *et al.*, 1993). Also PET has shown decreased glucose utilization in the medial–frontal regions of neurologically unaffected alcoholic patients (Samson *et al.*, 1986). Volkow *et al.* (1993) investigated the effect of lorazepam on regional brain glucose metabolism in 12 normal subjects and 10 alcoholic subjects with the use of PET and [^{18}F]fluorodeoxyglucose. They found that lorazepam decreased whole brain glucose metabolism in both the normal subjects (13%) and the alcoholic subjects (10% change), and the response was correlated with the concentration of lorazepam in occipital and cerebellar metabolism; the alcoholic subjects showed significantly less response than the comparison subjects in the thalamus, basal ganglia, and orbitofrontal cortex. The rate of response in the orbitofrontal cortex was significantly correlated with cerebellar metabolism at baseline. Adams *et al.* (1993) examined the behavioural correlates of medial–frontal lobe glucose hypometabolism in 31 chronically alcohol-dependent patients, assessed by PET and neuropsychological correlates [Wisconsin Card Sorting Test (WCST) and Halstead Category Test]. Results suggest that chronic alcohol intakes result in impaired function of cerebral tissue in the medial frontal region, affecting tissue metabolic rates and the behaviour correlates of these rates. Furthermore, Adams *et al.* (1995) investigated the correlation of neuropsychological function using the Wisconsin Card Sorting Test (WCST) and Halstead Category Test (HCT), with the rate of metabolism in subdivisions of the frontal lobes of older alcoholic patients measured with PET. They found that impaired performance on the summary subtest of the HCT was correlated with local cerebral metabolic rate for glucose in all three frontal subdivisions (cingulate, dorsolateral and orbitomedial), whereas the impairment in the summary WCST measure of categories was correlated only with local cerebral metabolic rate in the cingulate region. They suggested that these abnormalities in functioning of the subdivisions of the frontal lobe might contribute to different aspects of the behavioural impairment seen in older alcoholic patients. Additionally, Adams *et al.* (1998) evaluated the possible relationships between family history status and neuropsychological and neuroimaging results using PET. Forty-eight subjects, who had histories of severe chronic alcohol dependence, were divided into two groups: 27 with a first-degree relative with chronic alcoholism and 21 without a first-degree relative with chronic alcoholism. No differences were found between groups on either neuropsychological or neuroimaging tests. These results suggest that a family history of alcoholism does not moderate the damaging effects of severe chronic alcoholism on the functioning of the medial frontal lobe. However, Harden and Pihl (1995), in assessing the profile of cognitive dysfunction drawn from neuropsychological tests designed to assess the functional integrity of the frontal lobes, observed a relationship between high-risk status in sons of male multigenerational alcoholics and their performance on frontal lobe tests. Nevertheless the sample size was small (14 boys with, and 14 without, a positive family history of alcoholism).

Other recent studies (Deckel *et al.*, 1995; Gilman *et al.*, 1996) suggested that severe chronic alcoholism damages neurons containing $GABA_A$/benzodiazepine receptors in the superior medial aspects of the frontal lobes and that disturbance in integrity of the anterior neocortex may be a risk factor in the development of alcohol-related behaviours.

Single photon emission computed tomography (SPECT) studies

Techniques for measuring cerebral blood flow (CBF) have been available for about five decades. Initially, global CBF

was measured by determining the arteriovenous difference of the inert gas nitrous oxide. Later, the external detection of flow markers labelled with single photon emitting isotopes allowed more regional measurement of CBF. SPECT essentially measures regional cerebral blood flow (rCBF) by following the transport of a single photon-emitting radioisotope tracer to the brain and measuring the resultant activity with detectors. There are three main SPECT tracers for blood flow. ^{133}Xe, ^{99m}Tc-labelled hexamethylpropylene amine-oxine (HMPAO), and ^{123}I-labelled IMP (iodoamphetamine).

Early studies generally showed reduction in CBF in chronic alcoholics and patients with Korsakoff's syndrome (Meyer *et al.*, 1985; Ishikawa *et al.*, 1986). However, several studies suffered from serious shortcomings: the invasiveness of the procedures, usually involving catheterization of one carotid artery, made it difficult to have really normal control groups; also, patients were often on pharmacotherapy with sedating drugs, while being studied. In several interesting reports, CBF was measured by the non-invasive ^{133}Xe inhalation method in 222 volunteers recruited for a study of the effects of ageing, risks factors for cerebrovascular disease and dementia on CBF. Gray matter CBF significantly inversely correlated with the average alcohol consumption over previous years, which ranged from nil to heavy social drinking. This was regardless of whether or not risk factors for cerebrovascular disease were present (Roger *et al.*, 1983). Flow measurements were reported in patients with Wernicke–Korsakoff syndrome (Meyer *et al.*, 1985) and in chronic alcoholic patients without signs of Wernicke–Korsakoff syndrome (Ishikawa *et al.*, 1986). Both gray and white matter flow were reduced by ~20% in the Wernicke–Korsakoff patients. In chronic alcoholic subjects, values for gray matter flow were normal. Patients were restudied after several weeks of abstinence (and thiamine treatment in the case of the Wernicke–Korsakoff patients). In the compliant subgroups, flow values increased to normal in both sets of patients.

A French study looked at the relation between hepatic pathology and CBF in chronic alcoholics. Reductions in flow were found to correlate with the severity of the hepatic histological abnormalities, though not with the results of the usual biochemical liver function tests (Valmier *et al.*, 1986). These findings are in line with one of the first CBF studies in alcoholism where the results showed the greatest flow reduction in patients with cirrhosis (Shimojo *et al.*, 1967). Additionally, later pathological studies supported the contribution of alcohol, thiamine deficiency and cirrhosis of the liver to cerebral cortical damage in alcoholics (Harper and Kril, 1985; Kril, 1995).

Berglund and Risberg (1981) made serial bilateral measurements of regional rCBF by the ^{133}Xe inhalation method, during 13 withdrawal periods in 12 male alcoholics with pronounced physical dependence. A significant global reduction of rCBF was found during the first two days of withdrawal. Tutus *et al.* (1998) Performed [^{99m}Tc]HMPAO brain SPECT on the day of admission in non-medicated conditions and again after all the withdrawal symptoms had subsided in the patients. Results indicated that there were significantly reduced left frontal and right frontal, parietal and temporal rCBF values in the patients during alcohol withdrawal compared to those of their remitted state, which were not different from those in the control group.

Nicolas *et al.* (1993) studied the prevalence of central nervous system damage due to ethanol. They evaluated 40 asymptomatic chronic alcoholics and 20 age-matched controls. Studies included neuropsychological testing, brain [^{99m}Tc]HMPAO SPECT, and morphometric analysis by CT scan. In the quantitative analysis, 30 of the 40 alcoholics showed hypoperfusion areas on the SPECT scan. In the semiquantatitive analysis, alcoholics exhibited significant reduction in the rCBF ratio of all brain lobes, compared to controls ($P < 0.001$). The rCBF ratio was specially reduced in frontal lobes (by 65%).

Benson *et al.* (1996) described a case of acute alcoholic-induced Korsakoff amnesia in a 32-year-old female. SPECT showed hypo-perfusion in the orbital and medial–frontal lobe regions and the medial diencephalic area. They repeated the SPECT 4 months later. This showed a return to normal perfusion in the frontal brain areas, but little improvement in the medial diencephalic region. However, it is difficult to generalize from only one case report.

Kuruoglu *et al.* (1996) examined 40 patients with alcohol dependency, including 15 with antisocial personality disorder, as defined in DSM-III-R, and 10 age- and sex-matched healthy controls. The alcoholics were studied after termination of withdrawal symptoms, using high resolution SPECT, CT, and brain stem auditory evoked potentials (BAEP). The authors found a significant reduction in regional cerebral blood flow (rCBF) measurements of the alcoholic patients. Low flow in frontal regions encountered in 67.5% of the patients was associated with the duration of alcohol consumption, whereas no such relationship existed with the amount of daily intake. However, patients with antisocial personality exhibited more marked frontal hypo-perfusion.

Meanwhile, Jagannathan *et al.* (1996) assessed the brain metabolic changes in alcoholism by using localized proton magnetic resonance spectroscopy. They studied the brain metabolic changes in 10 alcoholic patients in the frontal lobe, cerebellum, and thalamus regions. The results obtained were characterized by a reduced *N*-acetyl-aspartate (NAA):choline (Cho) and NAA:total creatine ratios relative to age-matched ($n = 27$) controls. These decreased ratios correspond to depleted concentration of metabolite levels. Reduction of NAA is consistent with neuronal loss, whereas reduction in Cho suggests significant changes in the membrane lipids of alcoholics.

Interestingly, Gansler *et al.* (2000) examined the relationship between cerebral hypo-perfusion and residual deficits in the functioning of frontal brain systems in abstinent alcoholics long-term. CBF was observed through the use of SPECT perfusion images. Results showed a positive relationship between perfusion levels in the left inferior frontal brain region and years of sobriety. Alcoholics with less than 4 years of sobriety had significantly reduced left inferior frontal perfusion, compared with both non-alcoholic controls and alcoholics having longer periods of sobriety. The findings support the hypothesis that frontal brain abnormalities in alcoholics may subside with extended abstinence.

Additionally, both electroencephalographic (EEG) and evoked potential studies support the presence of neuro-physiological changes in brains of alcoholics, particularly in the frontal lobe (Pribram and Luria, 1973; Begleiter *et al.*, 1980; Porjesz *et al.*, 1980; Michael *et al.*, 1993; Bauer

et al., 1994; O'Connor *et al.*, 1994; Cohen *et al.*, 1996). These studies led Begleiter *et al.* (1980) to suggest that the presence of electrophysiological deficits even in the absence of apparent structural damage may possibly indicate the occurrence of neurochemical or subtle morphological changes not readily detectable by CT scan. They speculated that these electrophysiological deficits might reflect the imminent onset of overt structural changes.

STRUCTURAL ABNORMALITIES IN THE FRONTAL LOBE SYSTEM IN ALCOHOLISM

Post-mortem studies

Courville (1955), on the basis of neuropathological studies, noted the presence of cortical atrophy, more marked in the frontal lobes, but often widespread, with ventricular enlargement and meningeal thickening. The microscopic picture was one of cell loss, architectural disruption of the cortical laminae, pigmentary degeneration and proliferation of glial elements. In his experience, this picture was sometimes accompanied by marked arteriosclerotic changes. This description was supported by other authors (Warner, 1934; Hecaen and Ajuriaguerra, 1956; Mancall, 1961).

The picture of cortical atrophy, particularly involving the frontal lobes, may occur alone or in combination with other lesions. Victor *et al.* (1971) noted macroscopic cortical atrophy in 27% of their 72 patients, who showed the periventricular gray-matter lesions characteristic of Wernicke–Korsakoff's encephalopathy. Cortical atrophy has also been described in combination with degeneration of the corpus callosum in cases of Marchiafava–Bignami syndrome (Jequier and Wildi, 1955; Delay *et al.*, 1960).

A quantitative neuropatholological necropsy study of 22 control and 22 chronic alcoholic subjects showed a statistically significant loss of brain tissue in the chronic alcoholic group. The loss of tissue appeared to be from the white matter of the cerebral hemispheres, rather than the cerebral cortex (Harper *et al.*, 1985). In addition, a quantitative neuropathological necropsy study of the human cerebral cortex showed that the number of cortical neurones in the superior frontal cortex in chronic alcoholic patients is significantly reduced compared with that in controls matched for age and sex (Harper *et al.*, 1987). An analysis of brain weights has demonstrated a decrease of mean values in male alcoholics, when compared with controls. This weight loss occurred independently of the presence of Wernicke's encephalopathy, indicating that alcohol consumption is more important than nutritional deficiency in causing a reduction in brain weight (Harper and Blumbergs, 1982). However, the loss of brain tissue in chronic alcoholic patients was later found to be more severe in those who had nutritional vitamin deficiencies or alcoholic liver damage (Harper and Kril, 1985; Kril, 1995).

It has been suggested that the loss of white matter could be caused by changes in hydration, mainly loss of water (Carlen and Wilkinson, 1980). Such a hypothesis was supported by reports of altered body water balance in relation to the ingestion of alcohol and during the withdrawal phase in alcoholics (Eisenhofer and Johnson, 1982). However, post-mortem studies of the specific gravity and water content of the white matter negate this hypothesis (Harper *et al.*, 1987, 1988*a*,*b*).

The neuropathological lesions encountered in chronic alcoholics are likely to be the end result of a variety of aetiological factors. Post-mortem studies, and perhaps more so those based on forensic autopsies, often suffer from poor ante-mortem documentation. Some of these studies tend to be based on groups of elderly patients who have long drinking histories and may well also suffer from dietary deficiencies. Reporting, inevitably, tends to be selective. The presence of cerebral atrophy in a proportion of these patients is clear, but it is difficult to extrapolate these findings to the alcoholic population as a whole.

Recently, a series of studies of the effect of alcohol on receptors in the frontal cortex has been completed (Volkow *et al.*, 1993; Dodd *et al.*, 1996; Freund and Anderson, 1996; Gilman *et al.*, 1996; Lewohl *et al.*, 1997; Marchesi *et al.*, 1997). These studies concluded that chronic alcoholism leads to moderate increases in the density of the *N*-methyl-D-aspartate (NMDA) subtype of glutamate receptors in the frontal cortex. This up-regulation may represent a stage of alcohol-induced chronic neurotoxicity.

Neuroradiological studies

Until the introduction of CT and MRI scanning, the method required for visualization of cerebral structures was the injection of air in the subarachnoid space (pneumoencephalogram, PEG). As this technique carries a certain morbidity and discomfort, its application is limited to those patients in whom clear clinical indicators are present, such as the need to exclude a space-occupying lesion (Ron, 1977).

Brewer and Perrett (1971) examined 33 alcohol problem patients, using PEG, admitted to a psychiatric hospital. The mean age of the patients was 50 years. It was concluded that cortical atrophy was present in 30 out of 33 patients and that the ventricles were enlarged in 24. Only two patients were regarded as having a normal PEG. Frontal atrophy appeared to be especially common and was noted in 28 patients of the 30 with cortical atrophy.

Numerous other studies have reported PEG changes in chronic alcoholics with cortical and subcortical atrophy, often involving frontal lobes (Tumarkin *et al.*, 1955; Lafon *et al.*, 1956; Lerebouillet *et al.*, 1956; Postel and Cossa, 1956; Ledesma Jimeno, 1958; Riboldi and Garavaglia, 1966; Haug, 1968; Carlsson *et al.*, 1970; Ferrer *et al.*, 1970; Iivanainen, 1975).

Some of the difficulties in evaluating PEG studies could be overcome if adequate control groups were available. To find a group of healthy volunteers is practically and ethically impossible. A different approach is to use groups of psychiatric patients as controls (Haug, 1968). Unfortunately, however, it is possible that a number of such patients might have some degree of brain damage and it would be inaccurate to equate their PEG appearance with those of normal controls. Furthermore, deviations from the norm in psychiatric patients may mask or minimize the abnormalities in the group under scrutiny. On the other hand, if the differences between the two groups are clear their significance is probably greater (Ron, 1977).

As the morbidity of PEG has restricted its usage, the advent of CT scanning and MRI has made it possible for the first time to study large and carefully selected groups of alcoholics, to repeat the investigation after a follow-up period, and to examine a normal population for comparison.

Fox *et al.* (1976) used the CT scan to study hospitalized alcoholic patients. They reported significantly increased ventricular size in alcoholic patients when compared to normal controls. Carlen *et al.* (1976) reported that all of the alcoholic patients they studied demonstrated neuroradiological evidence for cortical atrophy. Epstein *et al.* (1977) conducted CT examination in a group of 46 alcoholics and found that 61.4% showed evidence of cortical atrophy. Myrhed *et al.* (1976) and Bergman *et al.* (1980) found cortical atrophy in their alcoholic patients. Sixty per cent of these patients showed 'clear cut' to 'high grade' brain damage, whereas 8% showed none. Ninety five per cent had widened parietal sulci, and 69% of these also had widened sulci in the frontal locations. Cala *et al.* (1978) observed cortical atrophy in 73% of their sample. They noted that enlargement of cortical sulci was most prominent in the frontal and parietal areas. Ron (1977) found that 65% of his sample of alcoholics showed evidence of brain damage. Wilkinson and Carlen (1980) have also noted a high incidence of cortical atrophy among alcoholic patients. Ron *et al.* (1980) performed CT in 100 alcoholics and 41 controls. In their series, radiological abnormalities were detected in a considerable proportion of chronic alcoholics, when compared with normal controls. The radiological abnormalities extended both to cortical structures and to the ventricular system. However, regional variations did not appear to be very obvious. These latter authors suggested that, with the current lack of knowledge of the underlying pathology, it is better to refer to them as 'brain shrinkage' rather than 'brain atrophy', which assumes more specific neuropathological lesions. Rosse *et al.* (1997) suggested that frontal lobe pathology is associated with negative symptoms in patients with chronic alcoholism. The authors examined 19 chronic alcoholic in-patients (aged 18–60 years) in an alcohol treatment unit and found a significant relationship between severity of frontal atrophy measured by CT and negative symptoms, measured by the Scale for the Assessment of Negative Symptoms.

The contribution of gender and drinking history to CT brain changes in alcoholics was studied. CT scan studies of male alcoholics have revealed larger ventricles, and wider cerebral sulci and fissures, compared with control groups (Bergman *et al.*, 1980; Ron, 1983). Similar findings have been demonstrated in a controlled study of female alcoholics, who showed an equivalent pattern of CT brain scan abnormalities, but after a shorter period of excessive drinking and lower estimated peak alcohol consumption than reported in studies with male alcoholics (Jacobson, 1986). Consecutive series of male and female alcoholics, Alcoholic Anonymous (AA) members and controls were examined by interview and with a CT brain scan. The CT scan findings persisted after accounting for body weight and after matching for age and length of drinking history. The CT scan parameters of female AA members approached control values more completely and after briefer abstinence than did those of male AA members. These findings are consistent with sex differences in the vulnerability of the brain to alcohol toxicity, and its recovery with abstinence (Jacobson, 1986).

MRI studies supported the CT scan findings on the effect of alcohol on frontal lobe volume. Pfefferbaum *et al.* (1997) used MRI to quantify the extent and pattern of tissue volume deficit and cerebrospinal fluid volume enlargement in younger, versus older, chronic alcoholics and relative to normal controls. They divided their group of 62 alcoholic men into a younger group ($n = 33$, mean age 37.5 years, range 26–44) and an older group ($n = 29$, mean age 52.7 years, range 45–63) to examine whether, in addition to extent, the two age groups differed in pattern of tissue type and regional brain volume abnormalities quantified with MRI. The younger group had significant cortical gray, but not white, matter volume deficits and sulcal and ventricular enlargement, relative to age-matched controls. The older group had volume deficits in both cortical gray and white matters and sulcal and ventricular enlargement that significantly exceeded the younger alcoholic group. An analysis of six cortical regions revealed that, although both age groups had gray matter volume deficits throughout the cortex, the older alcoholic group had a selectively more severe deficit in prefrontal gray matter, relative to the younger alcoholic group. Similarly, the cortical white matter volume deficit in the older alcoholics was especially severe in prefrontal and frontal regions. The difference in brain dysmorphology between the two alcoholic groups cannot easily be attributed to potential alcohol history differences typically related to age, because the two groups had similar disease durations and amounts of lifetime alcohol consumption. These results provide evidence that the frontal lobes are especially vulnerable to chronic alcoholism in older men. In another study, Sullivan *et al.* (1996) used MRI to study the relationship between alcohol withdrawal seizures and temporal lobe white matter volume deficits. They examined 11 alcoholics who had experienced one or more alcohol-related seizures and 35 seizure-free alcoholics, relative to controls. Each alcoholic group showed significant bilateral volume deficits of the anterior hippocampus and frontal–parietal and temporal gray matter, relative to controls.

MRI parameters also provide information about brain water. Two studies in chronic alcoholism have produced contradictory results, one describing changes suggestive of decreased free water content during withdrawal (Besson *et al.*, 1981), the other reporting an increase in free water during chronic alcohol consumption with a decrease during withdrawal (Smith *et al.*, 1985). In both reports, patient numbers were small (six and nine patients respectively). Harper *et al.*'s (1988*a*,*b*) post-mortem studies of brain water and brain-specific gravity supported the findings of the second of the above two MRI reports.

The conclusion is therefore that the occurrence of morphological abnormalities in brains of chronic alcoholics who appear clinically intact has been recognized. Neuropathological changes have been described, neuroradiological studies have demonstrated abnormalities compatible with cerebral atrophy, and the changes have often been detectable. However, the relative roles of alcohol toxicity, thiamine deficiency and cirrhosis of the liver in the pathogenesis of alcohol-related brain damage are still unclear.

IMPAIRMENT ON NEUROPSYCHOLOGICAL TESTS OF FRONTAL LOBES IN ALCOHOLISM

Although general measures of intelligence, especially those with a large verbal component, do not reveal deficits in performance in alcoholics (Tarter, 1975*b*, 1980; Parsons,

1987), detailed testing across different cultures has shown deficits in cognitive flexibility, problem solving, verbal and non-verbal abstraction, visuo-motor co-ordination, learning, conditioning, and memory (Jones, 1971; Jones and Parsons, 1972; Parsons, 1975, 1977; Tarter, 1976; Butters *et al.*, 1977; Cala *et al.*, 1978; Jenkins and Parsons, 1979; Tarter, 1980; Bergman, 1985; Miller, 1985; Acker, 1986; Wilkinson, 1987; Nicolas *et al.*, 1993; Beatty *et al.*, 1996; Nixon and Bowlby, 1996).

To detect lesions in the frontal lobes, standard intelligence tests tend to be of limited value. Special psychometric procedures may be more rewarding. Standard intelligence tests have nevertheless been used frequently in surveys of alcoholics, and their main contribution has been to demonstrate that there is no significant difference in IQ between large groups of chronic alcoholics and the normal population (Amark, 1951; Peters, 1956; Bauer and Johnson, 1957).

Attempts have been made to find a typical pattern of performance of chronic alcoholics in the subtests of the Wechsler Adult Intelligence Scale (WAIS). Wechsler (1941) studied a group of 29 chronic alcoholics of normal intelligence, aged from 36 to 55 years, and found that the subtests that offered most difficulty were the Similarities, Digit Symbol, Digit Span and Object Assembly. From this pattern of performance, he concluded that chronic alcoholics were relatively poor at abstract reasoning, perceptual organization, learning and retention. Goldstein and Shelly (1971) made a similar attempt.

More striking results have been obtained when tests especially designed to be sensitive to frontal lobe damage were used. Among these, the Porteus Maze test (PMT; Porteus, 1965) is worthy of mention. The test consists of a number of labyrinths, each increasing in difficulty. The subject must find the most direct route from beginning to end without entering blind alleys or crossing through lines. The subject must not lift the pencil while tracing the route and must keep within the side boundaries. As soon as an error is made, the same labyrinth is presented for a second trial, with a limit of two to four trials, depending on the degree of difficulty of the test. Two types of scores are obtained. A test age compares the particular achievement based on the number of repeated trials with a maximum score of 17, and a qualitative score assesses the type of production errors (e.g. cutting corners, touching the side). The Wisconsin Card Sort Test (WCST; Milner, 1963) requires subjects to learn the correct way to sort cards. On each trial, the subject must generate an attempt, sorting the cards according to the shape, colour, or number of symbols on it. The subject learns by trial and error on the basis of feedback provided by the tester. On this task, the subject does not learn a specific response, but rather a rule that governs their responses. Once they have learned to sort by one rule (such as sorting by shape), the rule is changed and the subject must sort by another rule (such as sorting by colour). When the subject has learned that rule, the rule is again changed, and so on until the subject has sorted all the cards.

The most widely used category identification test is the Halstead Category Test (HCT; Lezak, 1983), which consists of seven subtests, the first six measuring non-verbal abstraction-concept formation in which a series of slides are presented, each with different figures and patterns. The subject is instructed that something will suggest a number between 1 and 4; he then presses one of the four buttons and receives a feedback tone or buzzer telling him if the answer is correct. At the completion of each group, the examiner states that another group of items will be presented and the main idea may be the same or different. The seventh and final group demands memory of previous stimuli (Stuss and Benson, 1986).

Finally, two new batteries of tests have been prepared from the lifetime experience of the Russian neuropsychologist Luria. A selection of his tests has been translated into English by Christensen (1986), and a second, independent battery (Luria–Nebraska Neuropsychological Battery) designed to conform to current American psychological practices has been developed (Golden, 1986; Golden *et al.*, 1991). Theoretically, with the Luria approach comparatively subtle frontal lobe disturbance can be sought and isolated. In particular, subtests such as rhythm tapping, 'go/no-go', alternating figures, and others are suggested as particularly relevant to frontal lobe malformation.

Fitzhugh *et al.* (1960, 1965) studied a group of 35 alcoholics tested after an average of 12 days of abstinence and compared them with another group of 35 patients with well-documented brain damage and with 35 normal controls. The alcoholics were indistinguishable from the controls in terms of IQ. When the Trail Making test and Halstead Battery tests were used, the alcoholic group performed much worse than the control group and even worse than the patients with brain damage. Several other studies confirmed these findings and found evidence of frontal lobe damage by using the same tests or other frontal lobe tests (Jones and Parsons, 1971; Smith *et al.*, 1973; Long and McLachlan, 1974; Cutting, 1978; Hill, 1980; Goldstein and Shelly, 1982; Parsons, 1987; Sullivan *et al.*, 1993). However, Joice and Robins (1991) found that the impairment of alcoholic non-Korsakoff patients did not appear to be related to frontal lobe dysfunction, compared to Korsakoff patients. Non-Korsakoff patients exhibited fewer impairments that could not be attributed to deficits in either planning or spatial working memory.

Another important area of research was information processing. Previous research has demonstrated an impairment of information processing following alcohol administration (Koelega, 1995). However, it is not known whether this impairment is on all stages of information processing or on the early stages of it.

Studies examined different stages of information processing. The early stages of information processing have been described as those which involve the detection of, and response to, simple stimuli (Koelega, 1995). A task that assesses this function is the inspection time (IT) task, which involves the ability to make an observation/inspection of sensory input on which a discrimination of relative magnitude is based, in contrast to tasks such as reaction time (RT), which generally involve more response-oriented measures of total decision-making time, that constitute total information processing (Tzambazis and Stough, 2000). Several studies on the effect of alcohol on the early stages of information processing have found an impairment of speed of detection (Maylor *et al.*, 1990); attenuated auditory event-related potential (Jaaskelainen *et al.*, 1995); or increased reaction time and impaired stimulus detection (Krull *et al.*, 1994).

On the other hand, there has been considerable research into the effects of alcohol on total information processing, measured by RT, vigilance and attention tasks and tasks assessing cognition ability. Generally, results suggest that, when demands are higher, such as under dual-task conditions, the impairment in performance due to alcohol becomes more significant (Maylor *et al.*, 1990, 1992; Bartl *et al.*, 1996).

Tzambazis and Stough (2000) used IT as a predictor variable in a linear regression analysis to examine whether a disruption of the early stages of information processing accounted for changes in total information processing after alcohol administration. Results indicated that alcohol significantly slowed total information processing, independently of the early stages of information processing.

Acker (1986) assessed the contribution of gender and drinking history to neuropsychological deficits. Alcoholic inpatients were selected for cognitive assessment on a routine consecutive admission basis. The male and female alcoholic groups (72 males and 33 females) performed significantly worse on the cognitive tests than matched controls. The females performed worse on tests of immediate recall, and psychomotor speed. The author concluded that females are more susceptible than males to the harmful effects of prolonged heavy drinking on cognitive performance. Alternatively, it may be that those females who become alcoholic are 'at risk' in some unspecified way, which is reflected in the performance difference.

A large number of neuropsychological studies have examined individuals 'at risk' for developing alcoholism, such as sons of alcoholics (Schaeffer *et al.*, 1984; Drejer *et al.*, 1985; Tarter *et al.*, 1989; Peterson *et al.*, 1992; Knop *et al.*, 1993). They found that sons of alcoholic men exhibited increased impulsivity, decrements in attention, planning ability, and at-risk adolescents differed from low risk ones in Category test, memory tests, tests of abstracting/problem solving, Trail Making test, and significantly more errors on Halstead Category test and the Porteus Maze test. Collectively, these findings suggest that individuals at risk for alcoholism demonstrate a cluster of deficits that may somehow link anterior brain dysfunction to the risk of developing alcoholism.

Recently, it was reported that neuropsychological tests that assess anterior brain functioning are predictive of alcohol-related expectancies (Deckel *et al.*, 1995). Evidence of anterior neocortex involvement in the generation of these expectancies was also found through an examination of electrophysiological and structural data in some of these studies.

As mentioned above, Adams *et al.* (1993) studied the behaviour correlates of medial frontal lobe glucose hypometabolism in chronically alcohol-dependent patients. Thirty-one male patients who were detoxified, medically stable, and free of other central nervous system risk factors for neuropsychological impairment were examined with anatomical imaging (CT or MRI), functional imaging with PET and a battery of neuropsychological tests, including the Wisconsin Card Sorting Test and the Halstead Category Test. The findings suggested that chronic alcohol intake resulted in impaired function of cerebral tissue in the medial frontal region. The impairment pertains both to tissue metabolic rates and behavioural correlates of these rates. These findings have been supported by other studies (Bergman *et al.*, 1980; Ciesielski *et al.*, 1995; Deckel *et al.*, 1995; Nicolas *et al.*, 1997).

In summary, the impairment of neuropsychological function of the frontal lobe is particularly noticeable in the ability to perform complex psychomotor tasks and in some functions usually attributed to the frontal lobes, such as the ability to solve problems or to manipulate abstract concepts. These studies illustrate the fact that these deficits can be found in subjects whose IQ is average or even above average.

In everyday clinical practice, these tests for frontal lobe damage are seldom used and lesions in this area could easily be overlooked. Behavioural symptoms of frontal lobe impairment could readily be interpreted as part of the 'make-up' of the alcoholic's personality (Ron, 1977).

REVERSIBILITY OF FRONTAL LOBE CHANGES AFTER ABSTINENCE FROM ALCOHOL

The relationship between abstinence and recovery of frontal lobe deficits in chronic alcoholics is only partly understood. Adequate clarification would require following up a group of chronic alcoholics remaining abstinent for long periods, with careful repeat mapping of performance on appropriate investigations.

Page and Linden (1974) tested 20 hospitalized chronic alcoholics during the first week of abstinence. The WAIS, TMT and Benton Visual Retention tests were used. The authors observed an improvement in short-term memory, abstract reasoning, spatial ability, and visuo-motor co-ordination. By comparing successive performances the bulk of the improvement seemed to occur during the first 2 weeks of abstinence. This pattern of improvement was also found by Jonsson *et al.* (1962), Carlsson *et al.* (1973), Templer (1975) and Reed *et al.* (1992).

Clarke and Haughton (1975) studied a group of alcoholics at 2, 6 and 10 weeks after withdrawal: 130 patients were initially seen, but only 55 were tested three times. At psychological assessment, some improvement took place in the first 6 weeks of abstinence, but very little change was recorded thereafter. At 10 weeks, it was still possible to demonstrate impaired performance in psychomotor speed and abstract reasoning. In an attempt to rule out depression as a cause of poor test performance, the patients were asked to complete self-rating scales for depression at regular intervals. By the end of 6 weeks the patients rated themselves as 'normal', while psychological tests continued to show abnormalities.

The brain shrinkage was followed by re-scanning at 6 months to 3 years (Ron *et al.*, 1982; Ron, 1983). In the majority, appearances were unchanged, although some showed worsening, and an important group showed regression of the shrinkage. The factor clearly related to possibilities of improvement was the degree of abstinence achieved during the assessment period. These findings confirmed earlier ones (Carlen *et al.*, 1978; Carlen and Wilkinson, 1980). These findings were further checked in a group of patients drawn from AA (Jacobson, 1986; Lishman *et al.*, 1987). Despite earlier drinking histories, their scans were closer to those of normal controls than to those of current alcoholics; some degree of residual ventricular dilatation was suspected.

In summary, therefore, frontal lobe changes are potentially reversible to some degree with abstinence for several months or years, but even after several years the brain may remain abnormal.

GENERAL CONCLUSIONS

The occurrence of morphological abnormalities in the frontal lobes of chronic alcoholics who appear clinically 'intact' has been recognized. Neuropathological changes have been described, and neuroradiological studies have demonstrated abnormalities compatible with cerebral atrophy. The neuroradiological changes have occasionally been accompanied by cognitive deficits, detectable on careful psychometric testing, and were at least partly reversible.

Several PET and SPECT studies in alcoholics have already been reported and a clearer picture has begun to emerge. However, our understanding of the nature of the frontal lobe changes caused by alcohol, its clinical correlates, and its relation to prolonged drinking or abstinence, remains fragmented.

The possibility that neurophysiological, neuropathological, and neuropsychological changes may antedate the alcoholism is considered, as is the possibility that alcohol may be particularly damaging to the impaired brain. Evidence is accumulating that cognitive deficit is an important predictor of outcome following treatment. In the management of states of frontal lobe changes, attention should be paid to remedying nutritional deficiency and the general principles of rehabilitation should be borne in mind.

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Exhibit C

September 10, 2003

Mr. William Ray
Attorney at Law
5041 Airport Freeway
Ft. Worth, Texas 76117-6252

Mr. Timothy Moore
Attorney At Law
115 West 2nd Street, Suite 202
Ft. Worth, TX 76102

Re: **State of Texas v. Billy Jack Crutsinger; Cause No. 0885306D**

PRESENTENCE PSYCHOLOGICAL EVALUATION

Dear Mr. Ray and Mr. Moore:

I offer the report below in response to the requested evaluation of your client Billy Jack Crutsinger.

Referral Information:

Billy Jack Crutsinger is a 48 year-old divorced, Caucasian male who, after being charged with capital murder, was referred for evaluation by his defense attorney, William Ray. For this report, I was asked to conduct a detailed evaluation of the defendant's psychology and behavior at the time of the alleged offense, as well as all aspects of his history. The purpose of this evaluation was to assist the triers of fact in better assessing the following:

1. The defendant's background.
2. The defendant's character.
3. The defendant's personal moral culpability.
4. The defendant's mental state at the time of the offense.
5. What are the most important issues and factors to consider when attempting to predict a capital murder defendant's future dangerousness to society?
6. As best as can be determined, what is the defendant's overall estimated risk of future dangerousness? More specifically, is there a probability that he will commit acts of violence that would constitute a continuing threat to society?

Credentials:

My credentials as they pertain to this case are as follows:

1. I am a clinical forensic psychologist, licensed to practice in the state of Texas.

2. I am in private practice. My practice focuses solely on forensic cases (evaluation and treatment of individuals who have had, or are having, some interaction with the legal system).

3. I am the Chief Forensic Psychologist for the Behavior Management Treatment Unit of North Texas State Hospital – Vernon Campus, which is the only maximum-security forensic psychiatric hospital in the state of Texas. There, I treat the 50 - 60 individuals considered the state of Texas' most dangerous and violent psychiatric patients. As such, each month I must evaluate numerous individuals and make many clinical decisions concerning the level of dangerousness posed by those in my care.

4. I teach forensic psychology (psychology as it relates to the law) at the University of Texas at Dallas.

5. I have presented at both State and National conferences of forensic and mental health professionals concerning a variety of forensic issues, as well as the identification and management of dangerousness risk.

6. I have provided psychological evaluation, treatment, and programming consultation services to forensic institutions and professionals throughout the state of Texas, as well as in other states.

7. In the course of my training, employment with the State of Texas and my private practice as a Forensic Psychologist, I have evaluated over a thousand individuals.

8. I have worked for the prosecution, for the defense and for the court without concern for which "side" I work, as I provide my honest, clinical opinion regardless of who hires me.

9. A large percentage of my practice involves the psychological evaluation of individuals who have committed, or who are charged with committing, murder. Moreover, the evaluation of capital murder defendants comprises a goodly amount of my forensic practice.

10. My extensive forensic clinical experience provides me with a uniquely applicable knowledge base for forensic cases that is difficult to mimic or surpass in terms of the depth and breadth of my experience with unusual, damaged, heinous, dangerous, challenging or difficult to treat or evaluate individuals.

Notification:

Before beginning the evaluation, the defendant was informed of the purpose of this assessment and the confidentiality limitations as defined by law of any information obtained during this assessment. He was informed that an oral report would be provided to his attorney and a written report would be generated from this evaluation if his attorney so requested. He was advised that this examiner would provide any report written only to his attorney who could then dictate further distribution of said report. He was further advised that information from this evaluation could be used in court during the guilt/innocence and sentencing portions of his trial and could impact, either negatively or positively, any sentence assessed. The defendant indicated that he understood the purpose, nature and the inherent risks of this evaluation and he agreed to participate.

Evaluation Procedures:

The interviews, materials, and clinical procedures listed below were taken into consideration in the preparation of this report and the opinions contained herein. In order to accurately assess the defendant's background history, behavior patterns, emotional functioning, character, and usual mode of interacting with other's, third-party interviews were conducted with a variety of individuals and interviews were attempted with others who either refused to cooperate or could not be located.

A. Documents Reviewed

Please see Appendix A

B. Third-Parties Contacted

- Louise Crutsinger, the defendant's mother
- Pamela Fails Staples, the defendant's second wife
- Geraldine Suggett, the defendant's ex-girlfriend
- Ralph Crutsinger, the defendant's brother
- Darlene Glenn, the defendant's sister
- James R. Branton, friend of the defendant
- Donald E. Hall, friend of the defendant

C. Third-Party Interviews Attempted

- Linda Tucker Crutsinger, the defendant's third wife — Sent Letter, no response
- Susie Browder, the defendant's first wife — Unable to contact

D. Clinical Procedures, Instruments, and Measures

- Clinical interviews and testing with the defendant at Tarrant County Jail totaling approximately 14 hours on July 23 and 30 and August 8, 14 and 26 of this year. Some testing was conducted by Eric Miles, M.S. under Dr. Goodness' supervision
- The Adult Social History Self-Report Survey
- The Alcohol and Drug Use History Self-Report Survey
- The Birth History Self-Report Survey
- The Childhood Social History Self-Report Survey
- The Collateral Contact List
- The Criminal History – Self-Report Survey

- The Education History – Self-Report Survey
- The Employment and Military History – Self-Report Survey
- The Medical and Mental Health History – Self-Report Survey
- Personal Interests – Self-Report Survey
- The Personality Disorder Questionnaire IV (PDQ-IV)
- The Religious History – Self-Report Survey
- The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)
- The Sacks Sentence Completion Test (SSCT)
- The Tattoo and Scar Identification
- The Test of Memory Malingering (TOMM)
- The Trail Making Test – Part A and B (Trails A, Trails B)
- The Wechsler Adult Intelligence Scale – Third Edition (WAIS-III)
- The Wide Range Achievement Test – Revision 3 (WRAT-3)

Definitions

Mitigating evidence or mitigating factors - mitigating evidence or mitigating factors are any factors or evidence that any juror regards as reducing the defendant's moral blameworthiness and/or that militate against assessing the death penalty.

Triers of fact – the judge and/or jury in a legal case.

Preface

A detailed study of Billy Jack's psychology, the available records, and information garnered through collateral interviews has led me to develop a clinical formulation of the factors that likely contributed to Billy Jack's actions during the acts that ultimately caused the murders that he is charged with. Please note that the clinical formulation and information presented in this report are not intended to excuse, justify, or diminish the significance of any act that the defendant may have done, but are meant to help the triers of fact to understand why the defendant may have acted in such a manner. In particular, knowledge of the defendant's history and psychological make-up may assist the triers of fact in understanding where the defendant has come from and how he has come to be the man who is before the court today. When considered in its totality, this evaluation and report provide the triers of fact with detailed information concerning the defendant's background, character, personal moral culpability, the defendant's mental state at the time of the offense and his level of future dangerousness.

Current Offense

Basic Facts:

Eighty-nine year old Pearl Jordan Magouirk died after sustaining seven stab wounds to her torso and four cut wounds to her neck. A deceased seventy-one year old Patricia Magouirk Syren was found to have nine entry and one exit stab wounds of her neck, torso and arms. Ms. Magouirk had defensive cut wounds on her hands, including eleven cuts on her right palm, five cuts on the top of right hand and four cuts on her left hand. Their attacker took a car and a purse containing cash and credit cards before fleeing the scene of the murders.

Personal Background

Early Developmental and Social History:

Billy Jack was born on October 5, 1954 to Louise and Ralph Crutsinger. According to his mother, his birth was uncomplicated. Billy Jack was the youngest of four boys and two girls. Two of Billy Jack's brothers and one of his sister's were "handicapped." One brother had cerebral palsy and the other brother and sister are described as having been "slow," with the sister also having a "hard time walking." Those three siblings did not graduate from high school, but instead worked at a handicap center. Moreover, none of the Crutsinger children graduated from high school.

Billy Jack's father, Ralph Crutsinger supported his family by doing "road work," while his wife stayed at home with their six children. According to his wife, Ralph Crutsinger had a drinking problem that resulted in more than one DWI and some other legal problems. Likewise, alcohol also seemed to be the reason that Ralph Crutsinger would sometimes engage in domestic violence which was observed by the children. Ralph died in 1987 when Billy Jack was 27 years old.

Adult Social History:

Billy Jack first married when he was seventeen years old as his girlfriend, Susie Stuart, was pregnant. Billy Jack reported that he wanted to "do the right thing" and father the child; however, the child was born premature and died only a few hours after birth. The marriage did not last very long thereafter.

When Billy Jack was twenty-two years old, he married Pam Fails and the couple soon had a child they named Billy Earl. However, Billy Earl fell into a swimming pool at his maternal grandparent's house and drowned when he was a toddler. The couple later had another son, named Billy Jack, Jr., but he too died when he got into an automobile accident in 1994. Billy Jack Jr. was sixteen years of age at the time of his death. Even aside from the children's deaths, Billy Jack and Pam had a tumultuous relationship that ended in divorce.

Billy Jack went on to have several more significant romantic relationships. He married Sheryl Webb, but the couple only remained together for two years. He met Linda C. Tucker in 1989. That relationship was said to have been stormy with most arguments prior to their final separation being centered on Linda's son with whom Billy Jack had a poor relationship with. Linda and Billy Jack had multiple periods of separation. During several of his separations with Linda, Billy Jack had a relationship with Geraldine Suggett. According to Ms. Suggett, Billy Jack was never physically abusive to her, but he stole from her and lied to her. Ms. Suggett reported that Billy Jack was "a good person" when he was not drinking and that she continues to have some feelings for him, but knows that a relationship with him is extremely unhealthy for her.

Other landmark events in his life include a 1996 automobile accident in which Billy Jack was driving when another car hit him. His sister, Patsy, died as a result and Billy Jack is said to have taken her death hard. Not long afterwards, his brother, Randy also died.

Billy Jack's mother and his brother, Ralph, currently maintain contact with him through letters. Both individuals are in very poor health with Ralph having terminal cancer.

Educational History:

According to Billy Jack, he was considered "slow learning" and was in some special education classes. He reported that he failed the seventh grade one time, received remedial reading and math course work and dropped out of high school midway through the 11th grade. Records reflect that he had mostly average to slightly below average grades throughout junior and senior high school. Only partial educational records were provided to this examiner. These records did not indicate that Billy Jack was involved in special education programming. However, that may be because the records were incomplete and it is important to note that Billy Jack attended school before mandatory special education became law in 1974. Thus, he could have been provided special services without school officials feeling any special need to document their efforts as is now required. Given that Billy Jack has at least three siblings who required special education and none of his siblings graduated high school, and given that Billy Jack has current indicators of a Learning Disorder, it is probable that he received some special education.

Employment and Military History:

Billy Jack was never in the military, but has worked for a variety of construction and asphalt businesses and has also at times been a self-employed asphalt installer. Prior to the offense which occurred April 6, 2003, Billy Jack had been without work, aside from day labor work, for a number of months.

Mental Health History:

According to Billy Jack, his chiropractor referred him to a psychiatrist in 1997 for depression. He reported that he saw the psychiatrist approximately two times a week for three or four months and then less often for medication refills. He reported that his struggles with depression began after the automobile accident that took his sister's life. Billy Jack was also placed on an anti-depressant following his arrest for this offense and has remained on either Zoloft or Prozac since that time. He continues to report feelings of depression.

Medical History:

Billy Jack has a history of uncontrolled high blood pressure, gout, and he was diagnosed with alcoholic hepatitis. Billy Jack has had at least one incident of lost consciousness which was the result of hitting his head in an automobile accident in 1996.

Substance Abuse History:

Billy Jack began drinking alcohol at the age of seventeen. He favored beer and would sometimes spend eight or more hours at a time drinking in a bar. He twice received treatment for alcoholism in addition to attending Alcoholics Anonymous meetings. Both Billy Jack's father and his brother have received substance abuse treatment.

Legal History:

Billy Jack has served one year in a state jail. The first was for assault with bodily injury against his wife. The second was injury to the elderly which was an assault on his mother. He was placed on probation after both offenses and probation was eventually revoked after he failed

Three WAIS-III tables are listed below. The first contains the evaluee's age-corrected scaled scores. The second table contains their WAIS-III IQ scale data. Lastly, the third contains a summary of their WAIS-III index scores.

Verbal Tests	Scaled Scores		Performance Tests	Scaled Scores
Vocabulary	5		Picture Completion	11
Similarities	6		Digit Symbol-Coding	6
Arithmetic	4		Block Design	9
Digit Span	9		Matrix Reasoning	11
Information	5		Picture Arrangement	9
Comprehension	5		Symbol Search	4
Letter-Number Sequencing	8			

(Average = 10; Standard Deviation = 3)

WAIS-III IQ Scale	IQ Scores	95% Confidence Interval	Percentile	Qualitative Description
Verbal	74	70-80	4	Borderline
Performance	94	88-101	34	Average
Full Scale	81	77-85	10	Low Average

Difference between VIQ and PIQ = -20 ($p<.05$, Freq = 7.3%)

WAIS-III Index Scores	Index Scores	95% Confidence Interval	Percentile	Qualitative Description
Verbal Comprehension	74	69-81	4	Borderline
Perceptual Organization	101	94-108	53	Average
Working Memory	82	76-90	12	Low Average
Processing Speed	73	67-85	4	Borderline

On the WAIS-III, Billy Jack obtained a Verbal IQ (VIQ) score of 74 (4th percentile; Borderline), a Performance IQ (PIQ) score of 94 (34th percentile; Average), and a Full Scale IQ (FSIQ) score of 80 +/- 4 placing him in the Low Average range of intellectual functioning. The difference between his verbal reasoning IQ and nonverbal reasoning / performance IQ is statistically significant suggesting that his nonverbal reasoning skills are better developed than are his verbal reasoning skills.

Billy Jack also displayed an abnormal amount of subtest scatter on the Performance IQ scale in that less than 5% of people in the normal population display a comparable amount of performance subtest variability. Similarly, he displayed a wide degree of scatter across his total WAIS-III profile. Such scatter is seen in less that 15% of people in the normal population. Billy Jack's profile was indicative of an individual with a learning disorder. The fact that his spatial ability was better than his sequential ability combined with the fact that the sum of his Arithmetic, Digit Span, Information, Digit Symbol – Coding is suggestive of a learning disorder. Cognitive damage due to alcohol abuse is suggested in Billy Jack's relatively good visual organization skills versus his poor visual motor coordination. Long term alcohol abuse often directly affects visual motor coordination and processing speed.

In general, Billy Jack's verbal comprehension was poor as was his processing speed. In contrast, his non-verbal reasoning ability was a personal strength for Billy Jack as his perceptual organization score fell in the average range. His working memory was in the low average range.

His subtest scores on the R-BANS are listed in the table below:

R-BANS Subtest	Index Score	Percentile	Classification
Immediate Memory	76	10	Borderline
Visuospatial / Constructional	92	30	Average
Language	80	9	Low Average
Attention	88	21	Low Average
Delayed Memory	83	13	Low Average
TOTAL SCALE	**81**	**10**	**Low Average**

Billy Jack's performance on the R-BANS was best for visiospatial instructional activities. This corresponds with his performance on the WAIS-III. His worst performance on the R-BANS was on immediate memory tasks. However, his score did not differ significantly from his IQ. Likewise, Billy Jack's scores on language, attention and delayed memory tasks were in the low average range which are commiserate with his intellectual functioning.

His subtest scores on the Trail Making Test are listed in the table below:

Trails Part	Time *(in seconds)*	Errors
A	40	2
B	145	3

Billy Jack's test scores on the Trail Making Test suggest that he has some level of brain impairment.

Academic Functioning:

Billy Jack's scores on the WRAT-3 are listed below:

WRAT-III Subtests	Standard Score*	Percentile	Grade Equivalent
Reading	75	5	6
Spelling	66	1	4
Arithmetic	71	4	5

*(Average = 100; Standard Deviation = 15)

On the WRAT-3, Billy Jack's obtained scores are considerably below what would be expected given his attained grade level and age with his worst performance being on the Spelling subtest.

Testing Summary:

Billy Jack's overall performance on psychological testing indicated that he is not mentally retarded, but does have some intellectual and cognitive difficulties. It would be impossible to ferret out what portion of his deficits are the result of life-long learning and cognitive problems versus how much are due to his excessive alcohol use or even due to his uncontrolled high-blood pressure. Moreover, there is no particular definitive neuropsychological pattern exhibited by individuals who have damaged their brains due to alcohol use. What's more, Billy Jack has struggled with depression for the past several years. A number of his low scores could be related to the effects of the depression, but then again, they are also affected by alcohol abuse. For example, processing speed and attention are often affected with depression. When Billy Jack's test performance is considered in light of his past history of mediocre grades in school, possible special education, sub-standard employment, and alcohol abuse; they do not seem surprising.

In summary, Billy Jack's brain does not function in an altogether average manner. His ability to think with words, react with speed and use common sense is below average. However, his ability to visually take-in information, use that information to plan, as well as his analytic reasoning ability are in comparison good. Further neuropsychological testing may shed some additional light on Billy Jack's brain functioning deficits. However, consideration should be given to exactly how useful further clarification would be given the circumstances.

Diagnostic Impressions:

One question that this evaluation set out to answer was whether or not Billy Jack suffers from a mental illness or mental abnormality? The simple answer is yes. Based upon the data and testing outlined in this report, it is my opinion that the diagnoses listed below apply to Billy Jack.

Axis I: *Major Depressive Disorder*
Alcohol Dependence, in Institutional Remission
Learning Disorder NOS
Axis II: *Antisocial Personality Disorder*

1. *Major Depressive Disorder.* Billy Jack has a history of experiencing multiple symptoms of depression since approximately 1996. Billy Jack reported that at various times he has experienced a combination of the following symptoms: cries frequently, diminished pleasure, anxiety, suicidal ideation, irritability, memory disturbance, often tense, ruminates a great deal, frequently tired, insomnia and hopelessness. At least some of his mood problems may have been exacerbated by his alcohol use. He reported that he was prescribed antidepressants for his depression and is again on antidepressants.

2. *Alcohol Dependence, in Institutional Remission.* Billy Jack is an alcoholic. Research has shown that the risk of Alcohol Dependence is three to four times higher in close relatives of people with Alcohol Dependence.[1] Higher risk is associated with a greater number of affected relatives, closer genetic relationships, and the severity of the alcohol related problems in the affected relative. Thus, he likely has a biological predisposition to alcoholism as at a minimum, his father and brother were alcoholics. It is likely that his alcohol abuse has resulted in poorer brain functioning. Substance abuse problems are now less of a concern in Billy Jack's case, as accessing substances while incarcerated is costly and difficult.

3. *Learning Disorder NOS.* Billy Jack has a Learning Disorder that is most easily seen in his writing, spelling, and arithmetic skills. Billy Jack reported that he was previously labeled a "slow learner" and was given special education services. He failed at least the seventh grade. He has three siblings who were in special education and none of his five siblings graduated high school. Instead, they dropped out as did Billy Jack. Moreover, current testing indicates that his academic achievement scores are significantly lower than would be expected given his grade attainment and age.

4. *Antisocial Personality Disorder.* Billy Jack clearly meets the criteria for Antisocial Personality Disorder in that he has a pervasive pattern of disregard for, and violation of, the rights of others and has repeatedly broken the law.

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[1] Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (1994). American Psychiatric Association, Washington, D.C.

Clinical Formulation:

People are complex beings and the world is a complex place. Rarely is there a simple answer to the question of "why" someone did something. Most events, whether big or small, are usually the result of a combination of factors that have all come into play to produce the event. When conducting a comprehensive assessment for this type of case, it is important to attempt to identify and tease out the various situational, emotional, physical, medical, social, psychological, and environmental factors that combined to play a part in an event as tragic as the deaths of Patricia Syren and Pearl Magouirk.

It is my professional clinical opinion that the following factors combined to contribute to Billy Jack's actions during the index offense.

- *Alcoholism.* From the time Billy Jack was a young boy, he observed his father drinking beer rather than water even while working out in the sun. He thought that such behavior was normal and continued the tradition. Over the years, Billy Jack began to drink more and more beer and he became an alcoholic. Donald E. Hall, Billy Jack's longtime friend reported that he was once a bartender at a bar that Billy Jack frequented and that Billy Jack would sometimes spend Mr. Hall's entire eight hour shift drinking at the bar. Billy Jack's family members report that his drinking "got bad" approximately four years ago and Billy Jack began to be verbally aggressive and sometimes violent when he got drunk. His drinking must have been quite heavy as he was diagnosed with alcoholic hepatitis in 1996 following an automobile accident. Alcoholic hepatitis is a precursor to cirrhosis of the liver which comes from excessive, consistent alcohol consumption over a long period of time. He may well have progressed to cirrhosis now as seven years have passed since the hepatitis was diagnosed.

  When an individual has abused alcohol to the degree that their liver is affected, chances are that their brain will also have incurred damage. Furthermore, poor thinking and brain functioning occurs during times of intoxication. Those who remain constantly or consistently intoxicated will often exhibit faulty thinking and impaired judgment. Billy Jack's neuropsychological testing indicated that he has rather low scores across a number of domains tested. It is impossible to accurately discern the role that his alcohol abuse played in his low cognitive scores, but it is most likely that it did indeed have a role.

- *Lifelong lack of consequences.* Altogether, Billy Jack's life experiences consistently told him that he may sometimes have some consequences, but most of the time he could "get away with" acting on his impulses and desires as someone would be there to help him avoid the consequence's of his behavior. Billy Jack engaged in numerous behaviors throughout his life that would usually get an individual into serious trouble and which an individual would usually suffer major legal and social consequences, yet Billy Jack's behavior was met with relatively few meaningful consequences as he was often saved by other people from experiencing the consequences of his actions. His mother, his brother Ralph, his three wives, at least one girlfriend and some of his other friends are among the individuals who often bailed Billy Jack out of financial problems or legal scrapes. His biggest enabler seems to have been his mother, Louise Crutsinger, as she frequently

turned the other way when Billy Jack took her money or credit cards and she took up for him no matter what he did and would not allow him to "do without."

It seems that to some degree, even the legal system allowed Billy Jack to repeatedly act irresponsibly before taking action. For example, he finally did have his probation revoked from a 1993 offense that carried a suspended sentence; but this was only after he failed to attend required programs, failed to attend probation meetings, reported to probation meetings with alcohol on his breath, and failed to pay probation fees or court costs. Likewise, a 1998 offense carried a three year deferred adjudication sentence whose terms that Billy Jack consistently violated. He did not pay required court costs, fees or restitution. He did not keep counseling appointments or stay in the inpatient treatment program that he was referred to. He continued to drink daily during his probation and he did not attend anger management classes as required. However, eventually his probation was revoked and he spent a year in state jail for that offense.

- *Downward spiral.* Billy Jack had been spiraling downward much of his adult life. Three failed marriages, three dead children, a sister who died in an automobile accident in which he was driving, a brother who died shortly thereafter, frequent unemployment, financial problems, relationship problems, depression and legal scrapes were but some of Billy Jack's problems. These problems were often either a direct result of his alcohol use or were problems that were made worse by his alcoholism.

  Indeed, his downward spiral actually began in earnest when, much to his resentment, his mother could not tolerate his drinking anymore and left him to fend for himself. By that time, Billy Jack's drinking had escalated and he was beginning to be violent and aggressive when he drank. Once Billy Jack finally lost his biggest safety net, his mother Louise, he very much needed to find another safety net. He did so in several females, namely his wife Linda and a girlfriend named Geraldine Suggett; however, it was not long before Billy Jack burned out his goodwill with these ladies and was truly on his own.

  In December of 2002, Billy Jack's step-son got out of prison and began living with Billy Jack and his wife Linda. Billy Jack did not get along well with his stepson and much tension ensued. In January 2003 Billy Jack and his stepson began getting into physical altercations. In February, Billy Jack reported that he got into an argument with his wife about his stepson and his stepson again beat him up. That same month, Billy Jack's truck caught fire and burned due to a short in the wires and he was without transportation. By mid-March his home life had completely disintegrated. His wife gave him a bus ticket as she kicked him out of the house and he ended up in a motel in Fort Worth.

  Billy Jack did not have transportation and he had only four hundred dollars to his name. He began looking for work using taxis and public transportation. He was unsuccessful in his job search and the $400 did not last long. As a result, he began doing day labor in April. By May he was staying in a night shelter some nights and other nights he slept on the streets. Billy Jack reported that he "could not grasp what was going on. I used to be the one picking guys up for work and now I was the one being picked up."

Around the end of April or the beginning of May, he began staying in the Cowboy Motel on North Main. He felt very out of control. He had no money, no transportation, he felt abandoned by his friends and family and for the first extended period of time, he had no safety net and was completely alone. Billy Jack began to get desperate.

- *Alcoholic rage.* As should be self-evident, stressors increase the likelihood that individuals will act out aggressively. This is especially true of individuals prone to alcoholic rage as it seems Billy Jack is. Billy Jack's alcohol consumption had long been so great that many days, he would likely have continued to have had alcohol in his system the following day from what he consumed the night before. It is unknown if this were the case on the day of the offense, but at any rate, Billy Jack drank several beers before going to the victims' house in anticipation of securing a job that would get him on his feet. As he was growing increasingly more prone to do when drinking alcohol, Billy Jack went into a rage when he realized that Ms. Syren did not have a significant amount of work for him to do which meant that he would not be experiencing much stress relief. All of his anger at being left to fend for himself and of having his safety net taken from him was then brought to bear on the victims.

Future Dangerousness

The following questions were used to guide an assessment and discussion of the defendant's potential for future dangerousness.

- What are the most important issues and factors to consider when attempting to predict a capital murder defendant's future dangerousness to society?

- As best as can be determined, what is Billy Jack's overall estimated risk of future dangerousness? More specifically, is there a probability that he will commit acts of violence that would constitute a continuing threat to society?

Consistently accurate predictions of future instances of violence cannot be made; whether an individual will behave aggressively is a function of a *variety* of factors that include history, personal disposition, and situational variables (e.g., provocation, the setting) that cannot all be known in advance. However, it is possible to estimate relative risk by considering the individual's available historical data as research has identified a number of variables that are associated with either an increased or decreased risk. Among the most important factors that should be considered are the individual's prior violence, the context in which the violence occurred and the likelihood that a similar context will be encountered again in the future. This requires an understanding of the issues outlined below.

Setting

After working with many hundreds of the state of Texas' most dangerous and violent psychiatric patients, I have come to the conclusion that inaccurate predictions of dangerousness are at least in part due to a failure to take into consideration the setting for which the prediction is being made and a deep analysis of the context of previous violent acts. This is especially so given that the structure and boundaries provided by a particular type of environment vary from

setting to setting (e.g., prison vs. the free world), as do the behavioral expectations. Accurate predictions of dangerousness require that environmental factors be taken into account, as an individual may be a high risk of acting in a dangerousness manner in one environment, but a low risk in another.

If Billy Jack is found guilty of the current charges, there are only two possible placements: prison until he is at least 88 years old or death, in which case he would reside on Death Row in prison until the sentence was carried out. To consider his risk of dangerousness in any other setting besides prison would simply be wrong and would most likely lead to inaccurate conclusions about his true potential for dangerous behavior. Thus, for the purposes of maximizing the accuracy of the clinical predictions contained in this report, the defendant's potential future dangerousness is considered in the context of placement in prison, not in the free world community.

Context

Leaders in the field of psychology advise that serious mistakes will be made in assessing future dangerousness if the contextual approach is not used.[2] The basis of the current risk assessment considers Billy Jack's risk factors with particular attention to the contextual aspects of prior violent incidents, including the current offense, as doing so is vitally important to enhancing the accuracy of future dangerousness predictions.

Predictions of future dangerousness should take into account factors that have distinguished an individual's prior displays of aggressive behavior. Analysis of the individual's past violence can yield insights into themes that cut across the violent events and reveal person or situational factors that tend to be present during the individual's violent actions. A contextual analysis identifies the types of situations that has elicited violence from the individual, the method of their violence and their usual victim pool(s). This information can be compared to the likelihood that the individual will encounter similar violence triggers or opportunities for violence given the setting in which they will be placed. Furthermore, focusing on a behavioral analysis of the individual can uncover violence related factors not characteristically associated with violence in group studies (i.e., professional research).

Management and Control

Risk factors for future dangerousness must be considered in light of the intended setting's available risk management and control techniques.

Future Dangerousness Risk Assessment:

In my professional clinical opinion, the following issues and factors comprise the most important points to consider in assessing whether or not Billy Jack Crutsinger will present a future threat of danger to society.

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[2] Monahan, J., Steadmand, H,J., Silver, E., Appelbaum, P.S., Robbins, P.C., Mulvey, E.P., Roth, L.H., Grisso, T., & Banks, S. (2001). Testing "criminological" risk factors, in *Rethinking risk assessment: The MacArthur study of mental disorder and violence*, New York: Oxford University Press.

TDCJ Study

Recent research conducted with murderers confined to the Texas Department of Criminal Justice Institution Division identified six variables that were significantly related to violence among the incarcerated murderers.[3] This study is particularly applicable to capital murder cases in Texas as it was conducted with murderers in Texas.

- *Contemporaneous Robbery/Burglary.* According to this study of 6,390 murderers, involvement in a contemporaneous robbery/burglary at the time of the murder was associated with increased risk. Billy Jack did take some of the victim's belongings including money, credit cards and a vehicle. However, he did not do so in the course of the murder, but afterwards and he indicated that he did not originally intend to rob the victims when he entered the house. Thus, it is unclear if he actually meets this criterion as defined by this study, but I will assume that he does.

- *Multiple Victims.* The presence of multiple victims, as is true in this case, was found to be associated with increased risk.

- *Past Conviction of Attempted Murder or Assault.* A past conviction of attempted murder or assault were also found to be associated with increased risk. Billy Jack has had two assault cases which therefore increase his risk of dangerous behavior in prison.

- *Gang Membership.* The fourth variable that was associated with increased risk was gang membership in a prison gang. Billy Jack has never belonged to a gang and I do not believe he is likely to do so in the future. Thus, he does not possess this risk factor.

- *Prior Prison Term.* Having served a prior prison term was associated with increased risk. Billy Jack has served one year in a state jail.

- *Age.* The most influential indicator of prison violence in the Sorensen and Pilgrim study was the age of the defendant upon entry into the prison. Young offenders were most likely to engage in a violent act while older offenders were increasingly less likely to do so and their age became a protective factor that reduced an inmate's violence risk even if the inmate had all of the other risk factors that increased risk. Billy Jack is currently forty-eight years old and will soon be forty-nine. Thus, his age serves as a protective factor for his committing a violent offense in prison.

Other Relevant Variables

Significant history of nonviolent behavior over multiple incarcerations. Billy Jack's prior incarceration behavior can be seen as a very good indicator of his future incarceration behavior and strongly argues that Billy Jack is unlikely to be the one posing a danger in a prison environment. In the past, Billy Jack has been incarcerated in both county jails and state jail. Records from Jacksboro State Jail indicate that he was not a behavior problem there. His only behavioral report listed in the records provided this examiner indicates that he was cited for one failure to attend one class. Galveston County Jail records do not indicated that Billy Jack was a

[3] Sorensen, J. & Pilgrim, R. (2000). An actuarial risk assessment of violence posed by capital murder defendants. The Journal of Criminal Law and Criminology, 90(4), 1251-1270.

significant behavioral problem. The only negative notation in his records there indicated that he was noncompliant with jail staff members and medical treatment. However, the records do not specify if this means that he refused his antidepressant medication or exactly how he was noncompliant. Thus, it must have been a minor noncompliance issue. Likewise, Tarrant County Jail records indicate that Billy Jack is a low maintenance prisoner who has not posed a threat at any time during his incarceration there.

Past violence always accompanied by alcohol. It is important to an accurate future dangerousness prediction to understand that all of Billy Jack's previous aggressive behaviors have occurred during the course of using substances. The two earlier assaults that Billy Jack was convicted of both occurred while he was intoxicated. Billy Jack reported several physical altercations with his step-son in recent years. These also involved alcohol. Likewise, the current offense occurred after Billy Jack had consumed several bottles of beer. Remaining drug and alcohol free would decrease his risk while taking drugs and/or alcohol would greatly increase his risk. In fact, limiting Billy Jack's access to alcohol is in my profession opinion, the most important means of controlling his future violence risk potential. Without alcohol, Billy Jack is far less likely to have a rage reaction or to feel the type of artificial, false bravado and intensive emotion that often propels inebriated individuals into violent acts.

Violence usually directed toward women. Understanding an individual's usual victim of violence is essential to an accurate prediction of future dangerousness. Moreover, as previously mentioned, the context for which a future dangerousness prediction is made is of vital importance. Billy Jack's usual victim is a female (often older) who is either alone or in the company of another female. The two earlier assaults that Billy Jack has been convicted of were against women. Likewise, his relationships with his wives are said to have contained violence and the victims of the current offense were women. He does have one incident listed in his legal history that is related to an assault on a man outside of a bar, but that assault occurred back in 1974 when Billy Jack would have been 19 years old and is less relevant than more recent behavior. Billy Jack's only possible placement is in an adult male prison where there will likely be some female guards, but male guards are more prevalent which will thereby help to control his access to his usual victim pool. It will also be helpful that Billy Jack's access to alcohol while in the presence of females will be greatly limited.

Antisocial personality disorder. Though Billy Jack clearly meets the criteria for Antisocial Personality Disorder, which generally increases dangerousness potential, this disorder has an average duration of 19 years from the first symptoms to the last.[4] In fact, this disorder can become less evident or remit as the individual grows older, particularly by the fourth decade of life.[5] Thus, the prognosis for this condition is relatively good and is especially good in this case, since Billy Jack is nearing 49 years of age. Moreover, this diagnosis is common throughout any prison system (estimates range to 80%) and these individuals usually adjust well to incarceration.

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[4] Meloy, R. (1995). Antisocial Personality Disorder in G. Gabbard, ed. *Treatments of Psychiatric Disorders, 2nd edition.* Washington, D.C., American Psychiatric Press.

[5] *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (1994). American Psychiatric Association, Washington, D.C.

Childhood history of violence. A history of serious aggressive behavior during adolescence and particularly childhood (prior to age 13) is one of the historical factors most highly correlated with aggression and violence recidivism. Billy Jack does not have such a history.

Considering all of the above factors, it is my professional opinion that Billy Jack poses an overall **low risk** for violent or aggressive behavior in a prison environment for the foreseeable future. More specifically, there is a **low probability** that Billy Jack will commit acts of violence that would constitute a continuing threat to society. Suppression and deterrence of the kinds of violence shown by Billy Jack can, and have been, effectively done through the external controls inherent in a prison environment. Moreover, many of Billy Jack's risk factors can be effectively controlled in a prison environment.

Summary

In summary, the preceding information includes a multitude of factors that the triers of fact may take into account when considering appropriate sentencing. Information was provided concerning the defendant's background, character, personal moral culpability, mental state at the time of the offense and level of dangerousness.

Special care was taken to fully elucidate the identifiable factors that converged to bring about this tragic outcome. However, please feel free to contact me should more explanation be needed or should you have concerns of any nature. I appreciate the opportunity to be of assistance and participate in the legal process with this case.

Respectfully submitted,

Kelly R. Goodness, Ph.D.
Clinical and Forensic Psychologist
License #3-1223

Appendix A

Case Material Received

State of Texas v. Billy Jack Crutsinger, Cause No. 0885306D

Date	Material
4/03	Galveston Police Department records
4/03	Fort Worth Police Department records
4/29/03	Deposition of April Dawn Syren
4/9/03	Deposition of Robert Lee Greer, Jr.
4/15/03	Deposition of Tammie Lynn Crole
4/10/03	Warrant of Arrest
4/9/03	Miranda Warning
6/24/03	Numerous State's motions
1991-2003	Police reports on prior offenses
2003	Numerous photos of evidence & crime scene
July 7-8, 2003	Galveston Photos
	John Peter Smith Hospital Records
3/16/74	Fort Worth Police Department Offense No. 74-C-214, Arrest No. 05089-90-91
6/83	MHMR of Tarrant County records for Defendant
12/14/00-10/26/01	Department of Criminal Justice records
7/03	Letter of Bad Acts
	Birdville ISC
	Court Rec. 0660308D
	Court Rec. 1974
	Court Rec. 21458
	Credit Card Receipts
	Fort Worth Police Department Report Nos. 00271977, 02380650, 03044200, 03052069, Supplement
	Galveston Police Department Release
	Galveston Sheriff Department Medical
	Galveston Sheriff Record
	Galveston D.A.
	Hill County Sheriff Record

Case Material Received – Continued

	Photo Spread
	Picture of Defendant & finger
	Salvation Army
	State Jail Records
	Statement Lain
	Statement Moffet
	Statement Rouse
	Statement White
	Tarrant County Jail Records
	Tarrant Outreach Records
	Tarrant Records
	Tarrant County Jail Hospital Records
	TCME Corrected Diagram
	TCME Diagram
	TCME Transmittal
	TCME
	Vehicle Inquiry Receipt
4/9/03	Billy Jack Crutsinger's interview with Hardy & McCaskill
	Pictures of 1996 Cadillac, black bag, dumpster, & Galveston photos
	911 Calls
	Arrest Warrant
	Autopsies
	Biological Sample
	Bureau of Identification
	Bus Ticket
	Call Log
	7 Grand Jury Indictments
	Case Report
	Chain of Custody
	Meadowbrook Church
	Credit Card Log
	Credit Card Receipts
	Crime Lab Evidence Rpt.
	Crime Scene Log